

Trial of Labor after Cesarean Delivery as an Independent Risk Factor for Intrapartum Asphyxia in Singleton Deliveries beyond 37 Weeks' Gestation

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Abstract

The aim of this study was to determine the factors associated with intrapartum asphyxia in singleton deliveries beyond 37 weeks' gestation. We reviewed the obstetric records of Japanese singleton deliveries after 37 weeks' gestation managed at Japanese Red Cross Katsushika Maternity Hospital from 2005 through 2010. Forty-nine cases were diagnosed as intrapartum asphyxia on the basis of an Apgar score <4 at 5 minutes or umbilical arterial pH <7.0 or both. Cases with umbilical arterial pH ≥ 7.1 and 1-minute Apgar score ≥ 7 were examined as controls (n=10,484). Logistic multivariate regression analysis showed that intrapartum asphyxia was independently associated with cases of trial of labor after cesarean delivery (adjusted odds ratio, 3.24; 95% confidence intervals, 1.0–11; $p=0.04$). Our findings may be encouraging for the counseling of patients regarding a possible attempt at trial of labor after cesarean delivery.

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Key words: intrapartum asphyxia, term, singleton delivery, trial of labor after cesarean delivery, failed vacuum extraction/forceps delivery

Introduction

Recent studies have shown that intrapartum asphyxia is not the most important cause of cerebral palsy, probably accounting for no more than 10% of all cases; however, severe oxygen shortage in the brain and trauma to the head during labor and delivery are well-established causes of cerebral palsy^{1–5}. For example, in 1992 the American College of Obstetricians and Gynecologists established 4 basic criteria for defining the relationship between

perinatal events and the subsequent development of cerebral palsy²: umbilical arterial pH <7.0, Apgar score <4 at 5 minutes, neonatal neurologic sequence, and multiorgan system dysfunction. As many as 65% of children with cerebral palsy are born at term⁵, and the rate of cerebral palsy has not decreased in developed countries over the past 30 years. Therefore, the aim of the present study was to determine the factors associated with intrapartum asphyxia in singleton deliveries beyond 37 weeks' gestation.

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Patients and Methods

We reviewed the obstetric records of Japanese singleton deliveries after 37 weeks' gestation managed at Japanese Red Cross Katsushika Maternity Hospital from 2005 through 2010. The gestational age of the pregnancies was established with ultrasonographic examination of the fetal crown-rump length at 9 to 11 weeks' gestation. Cases of chromosomal anomalies or intrauterine fetal death before labor were excluded. Forty-nine cases were diagnosed as intrapartum asphyxia on the basis of an Apgar score <4 at 5 minutes or an umbilical arterial pH <7.0 or both. In this study, cases with an umbilical arterial pH ≥ 7.1 and a 1-minute Apgar score ≥ 7 were examined as controls (n=10,484). Furthermore, potential factors associated with intrapartum asphyxia were selected according to previous studies¹⁻⁷: maternal age, parity, gestational age at delivery ≥ 41 weeks' gestation, hypertensive disorders, premature rupture of membranes, oligohydramnios (amniotic fluid index <5 cm), placental abruption, trial of labor after cesarean delivery (TOLAC), and neonatal birth weight. In this study, delivery modes such, as emergency cesarean delivery and vacuum extraction (VE)/forceps delivery (FD), were also examined. Severe cerebral palsy was defined according to some previous reports⁸.

Cases and controls were compared with Student's *t*-test for continuous variables and the χ^2 test for categorical variables. Crude odds ratios (ORs) and 95% confidence intervals (CIs) were also calculated. Statistical analyses were performed with the statistical software package SAS version 8.02 (SAS Institute, Cary, NC, USA). Variables used in the multivariate model were those that had shown statistical significance ($p < 0.05$) on univariate analysis toward association with increased risk of the developing intrapartum asphyxia. Logistic regression was then performed to identify the factors most strongly associated in a multivariate model with the development of intrapartum asphyxia.

Results

The incidence of intrapartum asphyxia beyond 37 weeks' gestation was significantly greater in cases with nulliparity (crude OR, 2.46; 95% CI, 1.3-4.6; $p < 0.01$), gestational age ≥ 41 weeks' gestation (crude OR, 3.23; 95% CI, 1.8-5.9; $p < 0.01$), placental abruption (crude OR, 13.9; 95% CI, 3.2-60; $p < 0.01$), TOLAC (crude OR, 4.42; 95% CI, 1.6-12; $p < 0.01$), neonatal birth weight <2,500 g (crude OR, 2.46; 95% CI, 1.2-5.3; $p < 0.01$), or neonatal birth weight $\geq 3,500$ g (crude OR, 2.38; 95% CI, 1.18-4.8; $p < 0.01$; **Table 1**). During this period, there were 2 cases of uterine rupture after prior cesarean delivery; fortunately, neither of these cases was complicated by intrapartum asphyxia. However, there were no relations with maternal age ≥ 35 years, hypertensive disorders, premature rupture of membranes, the presence of umbilical cord prolapse/fore-lying, transcervical balloon catheters use, or oxytocin use.

Neonatal birth weight did not differ significantly between patients with intrapartum asphyxia and those without (**Fig. 1**). Logistic multivariable regression analysis showed that intrapartum asphyxia was independently associated with cases of TOLAC (adjusted OR, 3.24; 95% CI, 1.0-11; $p = 0.04$).

The rate of emergency cesarean delivery or VE/FD or both in cases with intrapartum asphyxia was significantly higher than that in cases without intrapartum asphyxia (**Table 2**). Cases of cesarean delivery following failed VE/FD was more strongly associated with intrapartum asphyxia than were cases of cesarean delivery or VE/FD alone ($p < 0.01$). Of the 7 cases of cesarean delivery following failed VE/FD, 3 were cases of TOLAC at 37 to 39 weeks' gestation, 2 were cases of TOLAC at 41 weeks' gestation, 1 was a case of heavy-for-dates infant at 40 weeks' gestation, and 1 was a case complicated by hypertensive disorders at 38 weeks' gestation.

The incidence of intrapartum asphyxia did not differ significantly between patients with successful TOLAC and those with unsuccessful TOLAC ($p = 0.06$; **Table 3**).

During the period, there were no patients with severe cerebral palsy.

Table 1 Obstetric and perinatal data for patients with and without intrapartum asphyxia in singleton deliveries beyond 37 weeks' gestation

	Intrapartum asphyxia	Control	P value
N	49	10,484	
Maternal age ≥35 years	21 (43%)	3,681 (35%)	0.26
Nulliparity	35 (71%)	5,185 (49%)	<0.01
Gestational age at delivery ≥41 weeks	15 (31%)	1,261 (12%)	<0.01
Hypertensive disorders	6 (12%)	665 (6.3%)	0.09
premature rupture of membranes	7 (14%)	2,107 (20%)	0.31
Oligohydramnios (amniotic fluid index <5 cm)	5 (10%)	751 (7.2%)	0.41
Placental abruption during labor	1 (2.0%)	32 (0.31%)	<0.01
TOLAC	6 (12%)	216 (2.1%)	<0.01
Umbilical cord prolapse/fore-lying	0 (0%)	17 (0.16%)	0.78
Neonatal birth weight			
<2,500 g	8 (16%)	771 (7.4%)	0.02
≥3,500 g	10 (20%)	1,020 (9.7%)	0.01
Transcervical balloon catheter use	4 (12%)	884 (8.4%)	0.95
Oxytocin use	19 (39%)	3,133 (30%)	0.18

TOLAC, trial of labor after cesarean delivery

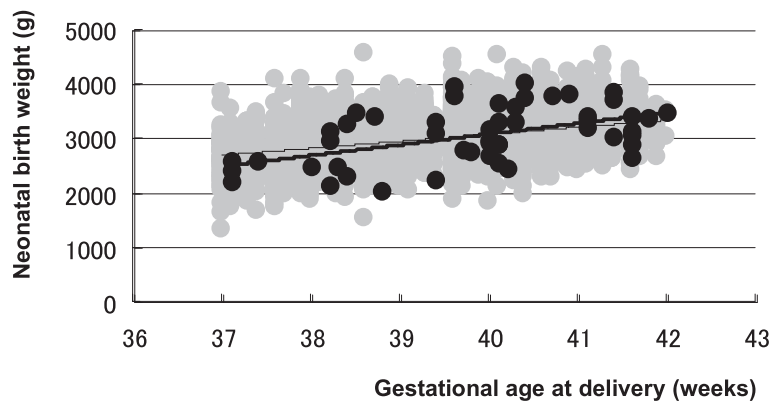


Fig. 1 Distribution of neonatal birth weight by gestational age at delivery with (black circles) and without intrapartum asphyxia (gray circles).

Table 2 Delivery modes of cases with and without intrapartum asphyxia in singleton deliveries beyond 37 weeks' gestation

	Intrapartum asphyxia	Control	P value
N	49	10,484	
VE/FD	14 (29%)	741 (7.1%)	<0.01
Cesarean delivery	10 (20%)	805 (7.7%)	<0.01
Cesarean delivery following failed VE/FD	7 (14%)	3 (0.029%)	<0.01
Vaginal breech delivery	0 (0%)	44 (0.42%)	0.65

VE/FD, vacuum extraction/forceps delivery

Discussion

The present study has found that TOLAC is an

independent risk factor for intrapartum asphyxia in singleton deliveries beyond 37 weeks' gestation. In this study, the incidence of intrapartum asphyxia did not differ significantly between patients with

Asphyxia Following TOLAC at Term

Table 3 Relationship between trial of labor after cesarean delivery and intrapartum asphyxia in singleton deliveries beyond 37 weeks' gestation

	Intrapartum asphyxia	Control	Odds ratio (95% confidence interval)
N	6	216	
Successful TOLAC	4	67	1.0
Unsuccessful TOLAC	2	149	4.45 (0.80–25)

TOLAC, trial of labor after cesarean delivery

successful TOLAC and those with unsuccessful TOLAC ($p=0.06$) because of the small sample size; however, intrapartum asphyxia may be associated with cesarean deliveries following failed VE/FD during TOLAC.

Several previous studies have found that the perinatal risk associated with TOLAC is low except for cases complicated by uterine rupture^{6,7}. For example, Landon et al.⁶ found that hypoxic-ischemic encephalopathy occurred in no infants born from 15,801 mothers who underwent elective cesarean delivery after previous cesarean delivery and in 12 infants born from 17,898 mothers undergoing TOLAC at term (0.067%, $p<0.01$). At their institution, symptomatic uterine rupture occurred in 124 women who underwent TOLAC (0.7%), and 7 of the 12 cases of hypoxic-ischemic encephalopathy, 2 of which resulted in neonatal death, were observed to follow uterine rupture (absolute risk: 0.046% in TOLAC). In addition, Socol et al.⁷ have observed that neonates with successful/unsuccessful TOLAC were not at increased risk for intrapartum asphyxia. In the present study, however, uterine rupture was not associated with intrapartum asphyxia. In addition, the present results indicate that nonreassuring fetal status might progress in cases of cesarean delivery, especially cases of TOLAC, following failed VE/FD. The present results also support our previous observation that cases of failed VE followed by cesarean delivery have a significantly higher incidence of neonatal complications than do cases of successful VE⁹. In cases of TOLAC, VE/FD may be more likely to fail than in cases without previous cesarean delivery because the Guidelines for Obstetric Practice in Japan of 2008 and 2011 have recommended limiting augmentation of labor pains during TOLAC¹⁰. The progression of nonreassuring

fetal status may be due to the time for preparations or anesthesia for cesarean delivery, because at our institution 10 to 20 minutes are needed to prepare for emergency cesarean delivery, which usually involves spinal anesthesia. Spinal anesthesia sometimes causes spinal hypotension associated with fetal acidemia¹¹. In addition, failed instrumental delivery, either VE or FD or both, in a setting in which cesarean delivery can follow promptly, has not been reported to be associated with increased morbidity of either the mother or the neonate^{12,13}. Therefore, the present results also reflect a serious problem at our institution, as reported previously⁹.

Although a much larger sample size would be needed to examine the perinatal risk of TOLAC at term, our findings are encouraging for the counseling of patients regarding a possible attempt at TOLAC.

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